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12/8/127787

doi:10.1067/mtc.2002.127787

### Reply to the Editor:

We appreciate the comments regarding our article by Oberwalder and colleagues and thank them for the valuable adjuncts regarding cerebrospinal fluid (CSF) drainage in stent-graft implantation.

Because of the restricted number of references in brief communications, we considered only publications in the high-impact journals. Nevertheless, we are aware of the report of Tiesenhausen and associates,<sup>1</sup> who described for the first time the impact of CSF drainage in a case of paraplegia after stent-graft implantation.

To close the entry tear in the descending aorta it is sometimes necessary to deploy a stent graft in the region (Th 8- L2) most prone to ischemia, but we agree that this should be avoided whenever possible. However, the risk of paraplegia has to be balanced against the risk of rupture of the aneurysm in these patients.

Routine use of CSF drainage in stent-graft implantations could help to reduce the risk of paraplegia in such cases.

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12/8/127793

### Alpha-stat strategy: Cause of ischemia in brains with old cerebral infarction despite selective cerebral hypothermic antegrade perfusion

#### To the Editor:

Washiyama and associates<sup>1</sup> are to be congratulated for experimentally verifying the anticipated susceptibility of brains with old infarction to become ischemic even during hypothermic selective continuous antegrade perfusion (SCP).

We do not doubt that SCP is better than no perfusion or retrograde perfusion, regardless of the pH management. However, despite SCP, ischemia developed in brains with old infarcts. Had the presented data been fully analyzed and interpreted without prejudice, the mechanisms of that ischemia would have been clear.

In our opinion, the alkalosis induced by alpha-stat hypothermic perfusion<sup>2</sup> explains every fact that the authors could not satisfactorily explain:

1. Although the authors failed to recognize and statistically analyze the increasing brain lactate efflux even in the control group from sample 1 to sample 4 during SCP, such increase seems to us significant. In our opinion, alpha-stat-induced alkalosis inhibits the creatine kinase catalyzed phosphorylation reactions.<sup>3</sup> This causes failure to aerobically synthesize high-energy~P bonds and switch to anaerobic metabolism with

consequent adenosine triphosphate consumption and lactate production even in the control group without prior infarct, despite the continuous perfusion.

2. Further lactate efflux increase in the infarct group after rewarming. Failure to use glucose aerobically becomes overt after rewarming following alpha-stat hypothermic uninterrupted perfusion at 20°C for 60 minutes.<sup>4</sup>

As pointed out by the authors, the penumbra area of an infarct is dependent on collateral flow. The vasoconstriction and decreased brain flow caused by alpha-stat strategies will certainly reduce such collateral flow, maximizing the metabolic effects of pH management. Sakamoto and associates<sup>5</sup> demonstrated brain anoxia development during early alpha-stat cooling before arrest. These effects are not limited to the period of cold perfusion but continue also during alpha-stat rewarming; thus, the lactate efflux at 32°C is maximal, even if circulatory arrest was not induced.

Obtaining the glutamate samples from the maxillary vein as often as the lactate samples would have answered whether the switch of aerobic to anaerobic metabolism and lactate efflux of the control group was enough to cause hypoxic excitotoxicity.<sup>6</sup>

pH-stat strategies increase brain blood flow and clearly should have been advantageous for perfusion of brains with old infarcts<sup>7</sup>; pH-stat hypothermic selective antegrade perfusion might prevent or minimize the metabolic effects of alpha-stat strategies even in brains with collateral flow-dependent penumbra areas.

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12/8/128323

doi:10.1067/mtc.2002.128323

### Reply to the Editor:

We would like to thank Miyamoto and Miyamoto for their insightful comments on our article. We have the following opinion in response to their observations.

Recent studies have revealed the advantages of pH-stat management over alpha-stat management in infants.<sup>1-3</sup> In the old cerebral infarct model that we used in our study, we also presume that pH-stat management might prevent the progress of anaerobic metabolism as indicated by Miyamoto and Miyamoto. However, we do not know whether pH-stat management may improve the surgical results in adult patients with old cerebral infarction, because atheromatous microembolism may also be as important a factor as ischemia of the penumbra. Although the metabolism in the penumbra that we described may be improved by pH-stat management, microembolism will certainly not decrease; if anything, it will increase.<sup>4</sup> Most of the adult patients who undergo aortic arch operations have atherosclerosis in the aorta, arch

vessels, and intracranial vessels. Patients with old cerebral infarction are especially likely to have severe atherosclerosis, whereas infants or young patients do not have any atherosclerosis. Moreover, blood flow of the penumbra would not necessarily increase in pH-stat management because there is a derangement of autoregulation in pH-stat management; this autoregulation is maintained in alpha-stat management.

We agree that pH-stat management has the potential of improving neurologic outcome in hypothermic operations. However, more investigations will be necessary to ascertain the actual merits of this strategy when applied in adult patients with severe atherosclerosis.

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12/8/128322

### Effects of storage solutions on in vitro vasoreactivity of radial artery conduits

#### To the Editor:

We read with great interest the work carried out by Chong and associates<sup>1</sup> on the

best solution for preservation of radial artery endothelial and contractile functions. We are not surprised to hear that the best solution was heparinized whole blood. For the past 4 years we have been harvesting radial arteries and intuitively believed that blood was the best solution. To this extent we have advocated that the radial artery should remain in the circulation until it is required as a conduit. Taking the radial artery out of the circulation and storing it in any form of storage solution will potentially make the endothelium ischemic, which in our view is the most significant factor in the development of spasm. We are curious whether the investigators have any additional information regarding the development of endothelial ischemia and degree of vasoreactivity.

Our experience in more than 1000 patients convinces us that maintaining the flow of blood through the artery is the most important factor in avoiding spasm. The radial artery can be dissected in the standard manner, after which the arm can be left out abducted between 80° and 90° or 10° and 30°. This provides enough space for the assistant to stand comfortably while the radial artery continues to be preserved in vivo by whole blood (heparinized or not). We do use topical papaverine or glyceryl trinitrate to counteract any spasm that may have developed during dissection. We have not found it necessary to use verapamil (topical or systemic) on any occasion to date.

We are delighted that Chong and associates have demonstrated the value of whole blood, as in our opinion it is the natural "preservation solution" for arteries normally. We would urge them and other surgeons to use our technique of keeping the radial artery in circulation for as long as possible before use.

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12/8/128074

doi:10.1067/mtc.2002.128074